

Subdural hematoma in Plasmodium vivax malaria: Another Nail hammered on the coffin

Sir,

The letter by Gosh *et al.*^[1] once again emphasizes that *Plasmodium vivax* infection is not always a simple and self-limiting infection. In comparison with *Plasmodium falciparum*, *P. vivax* runs a benign course and is rarely fatal. However, in recent times, scenarios have changed and there are reports of severe malaria due to *P. vivax*

infection.^[2] In this context, we would like to suggest the probable mechanisms based on our earlier observations^[3] and measures to tackle such situations.

Though the exact mechanism of subdural hemorrhage in this patient is not known, there is a possibility of rupture of small vessels plugged by infected red cells in combination with severe thrombocytopenia.^[4] This hypothesis has been supported by histopathological studies disclosing widespread cerebral vasculopathy due to parasite-specific factors such as adhesion and sequestration of parasitized erythrocytes in vascular endothelium with increased endothelial permeability, perivascular infiltrations, and cerebral edema; capillaries and venules are also distended and packed with erythrocytes.^[5]

In *P. vivax* infections, parasitemia seldom exceeds 2-5% of circulating RBCs, and high parasite indices are not attributed to the severity of the disease. However, cytokine production during *P. vivax* infections is higher than that during *P. falciparum* infections with similar degree of parasitemia.^[6] By up-regulating endothelial adhesion molecules, TNF- α may promote cerebral sequestration of platelets and red cells thus leading to hemorrhage. The serum TNF- α level correlates well with the severity of disease. In addition, patient-specific factors such as oxidative stress, enhanced host inflammatory responses, and alterations in splenic functions might have played a pivotal role. Thus, patient susceptibility to develop vascular complications may be attributable to gene polymorphism.

This is common for all clinical conditions and not specific for plasmodium vivax complications.

Subramanian Senthilkumaran, Chidambaram Ananth¹, Sandeep B. Gore², Ponniah Thirumalaikolundusubramanian³

Department of Emergency and Critical Care Medicine, Sri Gokulam Hospital, Salem, Tamil Nadu, 'Departments of Anaesthesiology and ³Internal Medicine, Chennai Medical College and Research Center, Irungalur, Trichy, ²Department of Accident and Emergency Medicine, Fortis Hospital, Mulund, Mumbai, Maharashtra, India

Correspondence:

Dr. Subramanian Senthilkumaran, Head, Department of Emergency and Critical Care Medicine, Sri Gokulam Hospital, Salem, Tamil Nadu - 636 005, India. E-mail: maniansenthil@yahoo.co.in

References

- Ghosh S. Curious association between Plasmodium vivax malaria and nontraumatic acute subdural hematoma. Indian J Crit Care Med 2014;18:335-6.
- Kochar DK, Das A, Kochar SK, Saxena V, Sirohi P, Garg S, et al. Severe Plasmodium vivax malaria: A report on serial cases from Bikaner in north western India. Am J Trop Med Hyg 2009;80:194-8.

- Senthil Kumaran S, Balamurugan N, Suresh P, Thirumalaikolundusubramanian P. Extradural hematoma in Plasmodium vivax malaria: Are we alert to detect? J Neurosci Rural Pract 2013;4 Suppl S1:145-6.
- Anstey NM, Russell B, Yeo TW, Price RN. The pathophysiology of vivax malaria. Trends Parasitol 2009;25:220-7.
- Menezes RG, Pant S, Kharoshah MA, Senthilkumaran S, Arun M, Nagesh KR, et al. Autopsy discoveries of death from malaria. Leg Med (Tokyo) 2012;14:111-5.
- Price RN, Tjitra E, Guerra CA, Yeung S, White NJ, Anstey NM. Vivax malaria: Neglected and not benign. Am J Trop Med Hyg 2007;77 (6 Suppl):79-87.

